CHRONIC TOXICITY SUMMARY

SULFURIC ACID

(dithionic acid; pyrosulphuric acid)

CAS Registry Number: 7664-93-9

I. Chronic Toxicity Summary

Inhalation reference exposure level	**1 µg/m^3**
Critical effect(s)	Bronchiolar epithelial hyperplasia, and thickening of the bronchial walls in monkeys
Hazard index target(s)	Respiratory system

II. Physical and Chemical Properties (HSDB, 1995; CRC, 1994; CARB, 1997)

Description	Colorless liquid
Molecular formula	H_2SO_4
Molecular weight	98.1 g/mol
Density	1.84 g/cm^3 @ 15° C
Boiling point	330±0.5°C (100%)
Melting point	10.36°C (100%)
Vapor pressure	<0.001 torr @ 25° C; 1 torr @ 145.8° C
Solubility	Soluble in water
Conversion factor	Not applicable

III. Major Uses or Sources

Sulfuric acid is a strong acid used as an intermediate in the synthesis of linear alkylbenzene sulfonation surfactants used in dyes, in petroleum refining, for the nitration of explosives, in the manufacture of nitrocellulose, in caprolactam manufacturing, as the electrolyte in lead-acid batteries, and as a drying agent for chlorine and nitric acid. Sulfuric acid is formed in the atmosphere from sulfur dioxide, from sulfur trioxide, and from oleum (a combination of sulfur trioxide and sulfuric acid used industrially). The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 4460 pounds of sulfuric acid (CARB, 1999).

IV. Effects of Human Exposures

Workers in the lead battery industry showed etching and erosion of the teeth after 4 months exposure to an average concentration of 0.23 mg/m^3 H_2SO_4 (Gamble *et al.*, 1984). Dental erosion increased in a dose-dependent manner with longer duration of exposure.

A study of 33 storage battery plant workers exposed to H_2SO_4 concentrations as high as 35 mg/m^3 showed a greater group mean decrease in FEV_1 across the time of their work shift compared to workers who were not exposed to sulfuric acid (El-Saddik *et al.*, 1972). The salivary pH of the sulfuric acid exposed workers, a qualitative measure of acid exposure, was lower than the controls during the course of the work shift.

OEHHA recently reviewed the California Ambient Air Quality Standard (CAAQS) for sulfates (25 $\mu g/m^3$ for 24 hours) to see if it adequately protects children (OEHHA, 2000). The report was peer-reviewed by the Air Quality Advisory Committee. The report indicates that H+ itself may play a role in the effects seen in epidemiological studies of sulfate air pollution. Controlled acute inhalation studies in humans and laboratory animals of pH neutral or nearly neutral sulfate salts (e.g., ammonium sulfate) (Utell et al., 1983; Lippman et al., 1987; Schlesinger et al., 1990), even at relatively high concentrations, do not produce the effects reported from epidemiologic studies of sulfates (asthma exacerbation, bronchoconstriction, decrements in lung function) that might be expected from short-term excursions. The controlled exposure studies show that sulfate aerosols containing strong acids, such as sulfuric acid and, to a lesser extent, ammonium bisulfate, produce functional and structural changes in healthy subjects consistent with those observed in epidemiological studies. A working hypothesis is that H^+ is a causal factor for adverse human health effects (e.g., see Lippmann and Thurston, 1996) and that, among the commonly measured particulate matter (PM) indices, $SO_4^=$ is the best surrogate metric for H^+.

A large number of epidemiologic studies have been conducted showing that elevated levels of several air pollutants, including acid aerosols, sulfur and nitrogen oxides, and particulate sulfates are correlated with an increased prevalence of pulmonary disease (U.S. EPA, 1989; OEHHA 2000). Elevated sulfate levels (1.6 ppb or 6.6 $\mu g/m^3$) have been associated with statistically significant decrements in FVC and FEV_1 in a cohort of Canadian children (Stern *et al.*, 1989). Further analysis of these data led Bates and Sitzo (1989) to conclude that H_2SO_4 was the most likely cause for the pulmonary changes observed. Similarly, Ostro *et al.* (1989) reported a statistical association between asthma-related symptoms reported by 209 asthmatics and sulfate and acidity levels in ambient air in Denver. Delfino et al. (1997) found that ambient H+ was associated with emergency room visits by children for respiratory symptoms in a study in Montreal. Additionally, Damokosh et al. (1993) in a follow-up analysis of the 6-City study suggested associations between average H+ concentration and chronic bronchitic symptoms. The relative odds of bronchitic symptoms with the highest acid concentration (58 $nmoles/m^3$ H+)_ versus the lowest concentration (16 $nmoles/m^3$) was 2.4 (95% CI:1.9 to 3.2). Furthermore in a study of children in 24 U.S. and Canadian communities (Dockery et al., 1996) in which the analysis was adjusted for the effects of gender, age, parental asthma, parental education, and parental allergies, bronchitic symptoms were confirmed to be significantly associated with strongly acidic PM (OR= 1.66; 95% CI 1.11-2.48). It was also found that FVC and FEV_1 were lower in locales with high particle acidity (Raizenne et al., 1996). Gwynn et al. (2000) reported an association between both H+ and sulfate particles and respiratory hospital admissions and mortality in Buffalo, NY. Acidic sulfates may act to increase the toxicity of particles by enhancing the availability of metals present in the particles to generate reactive oxygen species in the respiratory epithelium. This may account for some of the effects seen in these epidemiological studies and makes it difficult to use these studies as a basis for a Reference Exposure Level for sulfuric acid. The relationship between the effect levels observed in these

studies and the proposed REL is discussed in the section below on the potential for differential impacts on children's health.

The occupational standard for sulfuric acid is based on a study in human subjects by Amdur *et al*. (1952). In their study, 22 healthy male subjects were exposed to 0, 0.35, 0.4, 0.5, 1, 2, or 5 mg/m^3 for 5-15 minutes. The odor, taste, and irritation threshold was 1 mg/m^3. Since the basis for this standard is an acute exposure, it is not useful in determining a chronic non-cancer REL for sulfuric acid. A review of chronic human exposures to sulfuric acid and resulting carcinogenicity outcomes can be found in IARC (1992). However, none of the studies in that review examined non-cancer endpoints.

Sulfuric acid and oleum (supersaturated anhydrous sulfuric acid with varying concentrations of free sulfur trioxide) are absorbed as salts of sulfate anion (SO_4^{2-}), and are excreted as organic sulfates, neutral sulfur, or neutral sulfur compounds such as sulfur-containing amino acids. The low systemic toxicity of these metabolites is likely of secondary importance to the irritation caused by the inhaled acid.

V. Effects of Animal Exposures

An exposure of 9 cynomolgus monkeys per group to H_2SO_4 concentrations of 0, 0.38, 0.48, 2.43, and 4.79 mg/m^3 continuously for 78 weeks resulted in dose-dependent adverse histological changes in lung and bronchiolar epithelial and parenchymal tissue in addition to a dose-dependent decrease in blood oxygenation (Alarie *et al.*, 1973). In the animals exposed to 0.38 mg/m^3, significant bronchiolar epithelial hyperplasia was observed in 5 of 9 animals; thickening of the bronchiolar walls was observed in 3 of 9 animals. A slight focal bronchial epithelial hyperplasia was present in 4 of the 9 animals. One animal died after 4 weeks exposure to 0.38 mg/m^3. Although signs of pulmonary edema and cardiac hypertrophy were found, the cause of death was not determined.

Respiratory system effects of H_2SO_4 exposure in monkeys (Alarie *et al*., 1973)

H_2SO_4 (g/m^3)	*Particle size MMD*	*Bronchiolar epithelial hyperplasia Incidence – severity*	*Thickening of walls of respiratory bronchioles Incidence – severity*	*Increase in thickness of alveolar walls Incidence – severity*
0		0/9	0/9	0/9
0.38	2.15	5/8 – slight	3/8 - slight	0/8
0.48	0.54	0/8	0/8	0/8
2.43	3.60	8/8 – moderate	8/8 – moderate	8/8 – moderate
4.79	0.73	8/8 – moderate to severe	8/8 – moderate to severe	0/8

Alarie *et al.* (1973) also exposed groups of 50 guinea pigs of each sex to 0, 0.08, or 0.1 mg/m^3 H_2SO_4 continuously for 52 weeks. The group exposed to 0.1 mg/m^3 also received larger sized particulates than the 0.08 mg/m^3 group (2.78 μm vs. 0.84 μm, respectively). No exposure related effects were observed in the animals exposed to 0.08 mg/m^3, whereas exposure of 0.1 mg/m^3 resulted in decreased body weights in the female guinea pigs. No other histological changes in any organs were observed at the end of the 52-week study.

Rabbits (4 per group) were exposed to 250 μg/m^3 H_2SO_4 1 hour/day, 5 days/week for 4, 8, or 12 months. They showed significantly increased bronchoconstriction upon acetylcholine challenge after 8 and 12 months exposure, compared with a control group of 4 animals that received no H_2SO_4 (Gearhart and Schlesinger, 1986, 1988). Mucociliary clearance was also impaired by H_2SO_4 exposure and did not improve 3 months after cessation of exposure. A decline in dynamic lung compliance was observed after 12 months exposure. There was no evidence of inflammatory cell infiltration in the lungs of the exposed animals.

In guinea pigs, significantly slower and irregular breathing patterns were noted when the animals had inhaled albumin followed by 30 minute exposures to H_2SO_4 at 1.91 mg/m^3 twice per week for 5 weeks (Kitabatake *et al.*, 1979). Similarly, when guinea pigs were exposed to 2.49 mg H_2SO_4/m^3 for 4 hours/day, 6 days/week for 4 weeks, *in vitro* lung histamine release was significantly enhanced following heterogeneous albumin inhalation, compared to control animals unexposed to albumin (Fujisawa *et al.*, 1986; Iguchi *et al.*, 1986). In guinea pigs, sulfuric acid caused significantly greater lung function changes when adsorbed on the surface of zinc oxide particles as compared with pure sulfuric acid (Amdur and Chen, 1989). An exposure to 24 μg/m^3 sulfuric acid, layered on zinc oxide, produced significant reductions in lung function when followed by a brief exposure to 0.15 ppm ozone (Chen *et al.*, 1991).

A chronic exposure of beagle dogs to an average concentration of 889 μg/m^3 H_2SO_4 for 21 hours/day over a 620 day period resulted in increased expiratory resistance, reduced carbon monoxide diffusing capacity, reduced total and residual lung volume, and decreased lung and heart weights (Lewis *et al.*, 1973).

In apparent contrast to the above studies, rats and guinea pigs exposed to H_2SO_4 at 10 mg/m^3 for 6 hours/day, 5 days/week for 6 months exhibited no adverse histologic changes in lung tissue. Lung function measurements were not reported in this study (Cavender *et al.*, 1978).

Mice inhaled sulfuric acid mist at a concentration of 1.4 mg/m^3 in combination with a carbon particle mixture (1.5 mg/m^3) for 3 hours/day, 5 days/week for up to 20 weeks. The exposure resulted in significant alterations in specific antibody titer (decreased IgG, Ig_{2a}, IgM; increased IgG_{2b}), depression of primary splenic antibody response, and decreased resistance to respiratory infection as measured by mortality and survival time compared to controls (Fenters *et al.*, 1979).

There are no reliable studies indicating that sulfuric acid is a developmental or reproductive toxicant. In the absence of massive overexposure leading to maternal acidemia, H_2SO_4 will be neutralized in the maternal circulation and is unlikely to reach the fetus.

VI. Derivation of Chronic Reference Exposure Level

Study	Alarie *et al.*, 1973
Study population	Cynomolgus monkeys (5 males and 4 females per group or vice versa)
Exposure method	Continuous inhalation exposures (0, 380, 480, 2400, or 4800 $\mu g/m^3$) for 78 weeks
Critical effects	Significantly increased bronchial epithelial hyperplasia and bronchial thickening
LOAEL	380 $\mu g/m^3$
NOAEL	Not observed
Exposure continuity	The exposure was continuous during the experiment.
Exposure duration	78 weeks
Average experimental exposure	380 $\mu g/m^3$ for the LOAEL group
Human equivalent concentration	380 $\mu g/m^3$
LOAEL uncertainty factor	3 (slight effects)
Subchronic uncertainty factor	3
Interspecies uncertainty factor	3 (non-human primate)
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	300
Reference exposure level	1 $\mu g/m^3$

The study by Alarie *et al.* (1973) identified a LOAEL for chronic exposure to sulfuric acid of 380 $\mu g/m^3$. The principal uncertainties of this study are the small sample size of the test groups and the absence of an observed NOAEL. A lower chronic LOAEL for bronchial reactivity is presented by Gearhart and Schlesinger (1986, 1988) for rabbits (250 $\mu g/m^3$). This study was not selected as the basis of the REL because Gearhart and Schlesinger used only a single concentration of sulfuric acid, exposed the animals only for 1 hour per day for 5 days/week, used only 4 animals per group, and measured effects over the course of up to 12 months. The predominant weakness in the rabbit study, however, was the extreme discontinuity of the exposures (1 hour/day, 5 days/week), which would have necessitated use of a very large continuity adjustment. For these reasons, in addition to obvious physiological and genetic similarity arguments, the study in monkeys by Alarie *et al.* (1973) was felt to be more appropriate as the basis for the chronic REL for sulfuric acid. Alarie *et al.* (1975) determined a NOAEL for sulfuric acid in monkeys of 0.1 mg/m^3. However, other particulate matter (fly ash) was also present during the exposure. The Alarie *et al.* (1973) report provides data from exposure to sulfuric acid alone.

A free-standing NOAEL for histological changes in 100 guinea pigs exposed continuously for 1 year to 0.08 mg/m^3 was reported by Alarie *et al.* (1973). Guinea pigs respond to high concentrations of sulfuric acid by occasional laryngeal spasms that appear similar to a human asthmatic attack (Silbaugh *et al.*, 1981; Amdur and Chen, 1989). As a result, guinea pigs are thought to be sensitive models for the acute effects of sulfuric acid. For chronic effects of sulfuric acid on the lung, monkeys are likely a suitable model due to their physiological and structural similarites to humans.

For comparison, a chronic REL based on the guinea pig free-standing NOAEL of 0.08 mg/m^3 in animals exposed continuously for one year (Alarie *et al*., 1973) would be 0.8 μg/m^3.

VII. Data Strengths and Limitations for Development of the REL

The major strength of the study on sulfuric acid is the use of health effects observations from continuous long-term exposures to a primate. The major weaknesses are the lack of adequate human health effects data and the lack of a NOAEL observation.

VIII. Potential for Differential Impacts on Children's Health

There are no reliable studies indicating that sulfuric acid is a developmental or reproductive toxicant. Children are likely to be at greater risk from long-term exposures because their bodies are growing, and their developmental processes, especially in the lung, may well be impacted by air pollution exposures. Elevated sulfate levels (1.6 ppb or 6.6 μg/m^3) have been associated with statistically significant decrements in FVC and FEV_1 in a cohort of Canadian children (Stern *et al.*, 1989). The chronic REL for sulfuric acid of 1 μg/m^3 is below the level associated with those decrements in pulmonary function. However, in a study of moderately to severe asthmatic children (ages 7-13) (Thurston *et al*., 1997), a sensitive subpopulation for sulfate effects, approximately 1 μg/m^3 was the lowest level of ambient sulfate measured. The mean daily morning to afternoon peak airflow change, the use of beta-agonist medication, and the number of chest symptoms versus sulfate concentration in these children extrapolated linearly down to 1 μg/m^3. Thurston *et al*. (1997) also examined earlier data from Ontario (Burnett et al., 1994) on respiratory admissions to hospitals, and concluded that the sulfate threshold of effects, if it exists, lies below 5 μg/m^3, perhaps at about 2 μg/m^3. It should be noted that the sulfate and hydrogen ion effects are difficult to disentangle from each other and from the effcts of other PM constituents. The chronic REL of 1 μg/m^3 appears to have a relatively low margin of safety with respect to the epidemiological studies, but these observations are consistent with the proposed REL of 1 μg/m^3 since asthmatic children appear to be the critically sensitive human population for exposure to sulfuric acid (or sulfate).

IX. References

Alarie Y, Busey WM, Krumm AA, Ulrich CE, and Va V. 1973. Long-term continuous exposure to sulfuric acid mist in cynomolgus monkeys and guinea pigs. Arch. Environ. Health 27:16-24.

Alarie YC, Krumm AA, Busey WM, Urich CE, and Kantz RJ. 1975. Long-term exposure to sulfur dioxide, sulfuric acid mist, fly ash, and their mixtures. Results of studies in monkeys and guinea pigs. Arch. Environ. Health 30(5):254-262.

Amdur MO, and Chen LC. 1989. Furnace-generated acid aerosols: Speciation and pulmonary effects. Environ. Health Perspect. 79:147-150.

Amdur MO, Silverman L, and Drunker P. 1952. Inhalation of sulfuric acid mist by human subjects. Arch. Ind. Hyg. Occup. Med. 6:305-313.

Bates DV, and Sitzo R. 1989. The Ontario air pollution study: Identification of the causative agent. Environ. Health Perspect. 79:69-72.

Burnett RT, Dales RE, Raizenne ME, Krewski D, Summers PW, Roberts GR, Raad-Young M, Dann T, and Brook J. 1994. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. Environ Res 65:172-194.

CARB. 1997. California Air Resources Board. Toxic Air Contaminant Identification List Summaries.

CARB. 2000. California Air Resources Board. California Emissions Inventory Development and Reporting System (CEIDARS). Data from Data Base Year 1998. February 12, 2000.

CRC. 1994. CRC Handbook of Chemistry and Physics, 75th edition. Lide DR, ed. Boca Raton, FL: CRC Press Inc.

Cavender FL, Singh B, and Cockrell BY. 1978. The effects in rats and guinea pigs from six months exposures to sulfuric acid mist, ozone, and their combination. J. Environ. Pathol. Toxicol. 2:485-492.

Chen LC, Miller PD, Lam HF, Guty J, and Amdur MO. 1991. Sulfuric acid-layered ultrafine particles potentiate ozone-induced airway injury. J. Toxicol. Environ. Health 34:337-352.

Damokosh AI, Spengler JD, Dockery DW, Ware JH, and Speizer FE. 1993. Effects of acidic particles on respiratory symptoms in 7 US communities. Am Rev Respir Dis 147:A632.

Delfino RJ, Murphy-Moulton AM, Burnett RT, Brook JR, and Becklake MR. 1997. Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. Am J Respir Crit Care Med 155:568-576.

Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, and Speizer FE 1993. Health effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 104:500-505.

El-Saddik YM, Osman HA, and El-Gazzar RM. 1972. Exposure to sulfuric acid in manufacturing of storage batteries. J. Occup. Med. 14:224-226.

Fenters JD, Bradof JN, Aranyi C, Ketels K, Ehrlich R, and Gardner DE. 1979. Health effects of long-term inhalation of sulfuric acid mist-carbon particle mixtures. Environ. Res. 19:244-257.

Fujisawa T, Iguchi K, and Uchida Y. 1986. Effects of exposure to sulfuric acid mist on the induction of experimental asthma in guinea pigs. Japan J. Allergol. 35(2):137-144.

Gamble J, Jones W, Hancock J, and Meckstroth R. 1984. Epidemiologic environmental study of lead acid battery workers. III. Chronic effects of sulfuric acid on the respiratory system and teeth. Environ. Res. 35:30-52.

Gearhart JM, and Schlesinger RB. 1986. Sulfuric acid-induced airway hyperresponsiveness. Fundam. Appl. Toxicol. 7:681-689.

Gearhart JM, and Schlesinger RB. 1988. Response of the tracheobronchial mucociliary clearance system to repeated irritant exposure: effect of sulfuric acid mist on function and structure. Exp. Lung Res. 14:587-605.

Gwynn RC, Burnett RT, and Thurston GD. 2000. A time-series analysis of acidic particulate matter and daily mortality and morbidity in the Buffalo, New York region. Environ Health Perspect. 108:125-133.

HSDB. 1995. Hazardous Substances Data Bank. National Library of Medicine, Bethesda, MD (TOMES® CD-ROM Version). Denver, CO: Micromedex, Inc. (Edition expires 7/31/96).

IARC. 1992. International Agency for Research on Cancer. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 54. Occupational Exposure to Mists and Vapours from Strong Inorganic Acids and other Industrial Chemicals. Lyon, France: IARC, pp. 41-130

Iguchi K, Fujisawa T, and Uchida Y. 1986. Effects of exposure to sulfuric acid mist on the induction of experimental asthma in guinea pigs. Japan J. Allergol. 35(6):402-408.

Kitabatake M, Imai M, Kasama K, Kobayashi I, Tomita Y, and Yoshida K. 1979. Effects of air pollutants on the experimental induction of asthma attacks in guinea pigs: Sulfuric acid mist and mixture of the mist and sulfur dioxide. Mie Med. J. 29(1):29-37.

Lewis TR, Moorman WJ, Ludmann WF, and Campbell KI. 1973. Toxicity of long-term exposure to oxides of sulfur. Arch. Environ. Health 26:16-21.

Lippman M, Gearhart JM, Schlesinger RB. 1987. Basis for a particle size-selective TLV for sulfuric acid aerosols. Appl Ind Hyg 2:188-199.

Lippmann M, Thurston GD. 1996. Sulfate concentrations as an indicator of ambient particulate matter air pollution for health risk evaluations. J. Expo. Anal. Environ. Epidemiol. 6(2):123-146.

OEHHA. 2000. Office of Environmental Health Hazard Assessment. Staff report. Adequacy of California Ambient Air Quality Standards: Senate Bill No. 25 - Children's Environmental Health Protection.

Ostro B, Lipsett M, Wiener M, and Selner JC. 1989. A panel study of the effect of acid aerosols on asthmatics. J. Air Waste Management Assoc. 89-94.1:1-15.

Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, Ware JH, and Speizer FE. 1996. Health effects of acid aerosols on North American children: pulmonary function. Environ Health Perspect 104: 506-514.

Schlesinger RB, Chen LC, Finkelstein I, and Zelikoff JT. 1990. Comparative potency of inhaled acidic sulfates: speciation and the role of hydrogenion. Environ Res 52:210-224.

Silbaugh SA, Mauderly JL, Macken CA. 1981. Effects of sulfuric acid and nitrogen dioxide on airway responsiveness of the guinea pig. J. Toxicol. Environ. Health 8(1-2):31-45.

Stern B, Jones L, Raizenne M, Burnett R, Meranger JC, Franklin CA. 1989. Respiratory health effects associated with ambient sulfates and ozone in two rural Canadian communities. Environ. Res. 49(1):20-39.

Thurston GD, Lippmann M, Scott MB, Fine JM. 1997. Summertime haze air pollution and children with asthma. Am. J. Respir. Crit. Care Med. 155(2):654-660.

U.S. EPA. 1989. U.S.Environmental Protection Agency. An Acid Aerosols Issue Paper: Health Effects and Aerometrics. EPA/600/8-88/00SF. OHEA/ORD. Research Triangle Park, NC: U.S. EPA.

Utell MJ, Morrow PE, Speers DM, Darling J, Hyde RW. 1983. Airway responses to sulfate and sulfuric acid aerosols in asthmatics: an exposure-response relationship. Am Rev Resp Dis 128:444-450.

CHRONIC TOXICITY SUMMARY

VINYL ACETATE

(1-acetoxyethylene; acetic acid, vinyl ester; acetic acid, ethenyl ester; VAC; vinyl A monomer; ethenyl ethanoate)

CAS Registry Number: 108-05-4

I. Chronic Toxicity Summary

Inhalation reference exposure level	**200 µg/m^3** (50 ppb)
Critical effect(s)	Nasal epithelial lesions in rats and mice
Hazard index target(s)	Respiratory system

II. Physical and Chemical Properties (HSDB, 1994)

Description	Colorless liquid
Molecular formula	$C_4H_6O_2$
Molecular weight	86.09 g/mol
Density	0.932 g/cm^3 @ 20°C
Boiling point	72.7° C
Melting point	–93.2°C
Vapor pressure	115 torr @ 25°C
Solubility	Slightly soluble in water, soluble in ethane, acetone, chloroform; >10% soluble in ethanol and benzene
Conversion factor	1 ppm = 3.52 mg/m^3 @ 25°C

III. Major Uses and Sources

The major use of vinyl acetate monomer is in the manufacture of polyvinyl and vinyl acetate copolymers, which are used in water-based paints, adhesives, paper coatings, and applications not requiring service at extreme temperatures (HSDB, 1994). It is also used in safety glass interlayers and in hair sprays (HSDB, 1994). In the atmosphere vinyl acetate breakdown can result in formation of acetaldehyde. The annual statewide industrial emissions from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 3855 pounds of vinyl acetate (CARB, 2000).

IV. Effects of Human Exposures

Deese and Joyner (1969) conducted an occupational study of 21 chemical workers with a mean length of employment of 15.2 years and exposed to a time-weighted average of 8.6 ppm (30.3 mg/m^3) VA. No adverse effects were noted following chest x-ray, electrocardiogram, blood chemistry, and urinalysis. The control group (sample size unspecified) consisted of workers in